Rockwell (A.D.)

OBSERVATIONS

ON

HEMIPLEGIA,

BASED ON EIGHTY-ONE RECORDED CASES, WITH SPECIAL REFERENCE TO CEREBRAL LOCALIZATION.

BY

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ELECTRO-THERAPEUTIST TO THE N. Y. STATE WOMAN'S HOSPITAL, ETC.

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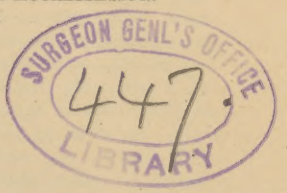

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OBSERVATIONS ON HEMIPLEGIA,

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TYPICAL CASE OF HEMIPLEGIA—LESIONS OF THE CORPUS STRIATUM—RELATIVE FREQUENCY OF RIGHT AND LEFT HEMIPLEGIA—ORGANIC APHASIA—FUNCTIONAL APHASIA—ACUTE SLOUGHING—INCREASED TEMPERATURE AND SWELLING OF PARALYZED MEMBERS—LESIONS OF THE OPTIC THALAMUS, CRUS CEREBRI, AND PONS VAROLII—"PULSUS DEFERENS"—LESIONS OF THE CORTICAL SUBSTANCE—IRREGULAR FORMS OF ANÆSTHESIA—UNILATERAL HYPERÆSTHESIA—SPASM OF VESSELS—INTERMITTENT HEMIPLEGIA—SUDDEN LOSS OF SIGHT, FOLLOWED BY RECOVERY.

I have records of eighty-one cases of hemiplegia that have been under my care during the past fourteen years, and it has occurred to me that some sort of an analysis of them might be not altogether valueless, both in a statistical point of view and as a clinical contribution to cerebral localization. The symptoms and the grouping of symptoms in many cases of hemiplegia vary very widely, and this variation, it is hardly necessary to say, is due to the special part or parts of the brain-substance that are damaged. Every disease and every pathological condition, however, has its common and readily recognized type, and the typical hemiplegic finds his counterpart in the following case:

Mr. X——, aged sixty-two, suddenly, and without marked premonitory symptoms, became partially unconscious, and on recovery found that his right leg and arm were completely paralyzed.

The face was drawn sharply toward the non-paralyzed side, and the tip of the tongue when protruded deviated to the paralyzed side. There was some aphasia, and the utterance was thick, but the intellect was undisturbed. Some anæsthesia of the extremities was present, but the electro-muscular irritability was normal. In the course of two weeks the patient was able to articulate with almost his usual clearness, and the aphasia had entirely disappeared. He could take a few steps around the room, while the paralysis of the face and tongue had improved in a considerably greater degree. In three months' time the patient was able to walk about at will, although the toe pointed somewhat downward, with a tendency to drag. The characteristic and almost pathognomonic circumduction of the leg had, however, almost entirely disappeared. As is usual in these cases, the arm recovered much less rapidly than the leg, and it was many months before he could raise it over his head or utilize it in any considerable degree. The regional or anatomical diagnosis in this case was effusion of moderate quantity into or in the immediate neighborhood of the corpus striatum. This patient finally so far recovered that he could walk with hardly a perceptible limp, while the facial muscles and tongue were quite restored to their normal condition.

The arm, however, remained decidedly weaker than its fellow until the occurrence of death, some two years subsequently—from a second and more profuse hemorrhage. In addition to this case, I find a sufficiently detailed record of eighteen others, the symptoms of which were enough in accord with the one just given as to justify a similar diagnosis as to the probable seat of the lesion. In thirteen of these nineteen cases the paralysis was on the right side, and in the remaining six upon the left side. Of these thirteen cases, involving lesions of the left hemisphere, nine were in a greater or less degree amnesic or aphasic, while of the six cases involving lesions of the right hemisphere, not one was complicated with aphasia or its kindred symptoms.

This experience is in accord with the generally

accepted view that aphasic symptoms follow lesions of the left cerebral hemisphere rather than the right, and that the posterior portion of the third convolution is the most frequent seat of structural change.

In analyzing all my cases—81 in number—with reference to the side affected, I find that right hemiplegia preponderated in about the same proportion as in the 19 typical cases (lesions in or about the corpus striatum) just referred to. Right hemiplegia occurred 49 times; left hemiplegia, 30 times, and double hemiplegia twice. While it is evident that complete and persistent hemiplegia with aphasic symptoms depends upon positive lesion of the left hemisphere, as a matter of fact, the aphasia very frequently disappears, almost entirely, while yet the paralysis of motion remains. This could not well be explained if it were true, as M. Broca long ago declared that the faculty of articulate language was located in the third left frontal convolution. On the contrary, the experiments of Fritsch, Hitzig, and Ferrier show that in every essential point the brain is symmetrical.

It is rational to believe, therefore, that the reason why loss of the co-ordinating power of speech is so universally associated with left cerebral lesion is, that as most people are right-handed, so are they left-brained, and in lesions of the left hemisphere with aphasic symptoms the memory of words is not totally destroyed, but the victim is without the power of articulate speech, because the right side of the brain has been so little exercised as to be incapable of at once acting alone. When aphasic symptoms disappear, therefore, it may be said that the right side of the brain becomes educated, in the same way that the left hand is educated when its fellow becomes disabled.

In regard to the side of the lesion in aphasia, it may be remarked that Trousseau, in a most extensive hospital experience, met with but one case of left hemiplegia associated with aphasic symptoms, which, at the time, he asserted to be the only one on record. It would be interesting if it were known whether Trousseau's case was left-handed as well.

Aphasia may undoubtedly occur without coincident paralysis, and dependent upon no serious structural changes. The single case of this character that has come under my observation occurred during convalescence from a severe attack of typhoid fever. The patient first noticed a slight impairment of the co-ordinating power of expression, which day by day increased in degree until he lost almost completely the power of intelligible converse. Under a general tonic treatment this patient finally recovered.

Slight disturbances of speech, unassociated with paralysis, generally disappear rapidly and without treatment, and if, as it is possible, a local change takes place, it can hardly be more than some slight so-called molecular disturbance. Following a somewhat graver central lesion, we have aphasia associated with paralysis, more or less persistent, but amenable to a greater or less extent to time and treatment.

As illustrating this condition, I give the following case, in which the treatment adopted seemed to be immediately and positively beneficial.

I was consulted December 7, 1873, by a lawyer, aged fifty-three. One morning, about eight months prior, he arose in his usual health, and for some hours attended closely in his office to a press of business that had accumulated during a few days' absence from the city. While thus engaged, he observed that his right leg was a little numb, and that there was a disagreeable sensation of tingling in the fingers of the right hand.

Almost immediately he felt that his whole right side was paralyzed, and on attempting to speak, found that he could not do so intelligibly. In a few days the patient regained almost the normal power over the paralyzed members, but his speech returned more slowly, and for three months before I saw him there had been little progress.

He could speak short, disconnected sentences well enough, but when he attempted to engage in animated conversation, he not only constantly used words that utterly failed to convey his ideas, but frequently, all remembrance and power of expression

seemed to forsake him, so that he was unable to proceed until reminded of the proper word, and the thought that he had been expressing.

The patient was treated for about a month by central galvanization, and with such marked benefit that in a few weeks he was able to converse with much greater readiness.

While aphasia is a result peculiar to injuries to the left, it is claimed that there are certain symptoms peculiar to injuries of the right cerebrum. Tonic spasms of the limbs and conjugated deviation of the eyes, are said to occur in the proportion of about two-thirds of the cases for left hemiplegia, and it has been found by Brown-Séquard that the same proportion of the "recorded cases of bed-sores, or acute sloughing in cases of hemiplegia, have occurred where the paralysis of the limbs has been on the left side."* My experience tends to confirm this statement, since of two cases of acute sloughing that I have seen, both were associated with lesions of the right hemisphere.

This symptom is regarded as indicative of speedy dissolution, and is believed to depend on trophic rather than on vaso-motor changes. In my own cases the patients died within a few weeks of the appearance of the sloughing.

In a certain rather limited proportion of cases of hemiplegia, we find that the paralyzed limbs become hot and swollen, and remain in this condition for an indefinite length of time. Out of the whole number of my cases, these symptoms were associated with five, and were especially marked in the following:

The patient, a man aged sixty, became suddenly numb throughout his right side, but speedily recovered. Soon after the attack was repeated, and with a similar result. The third attack resulted in complete paralysis of the right side, followed by swelling and increased temperature.

Six months subsequently when I saw him the symptoms were typical of an ordinary lesion of the

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* Bastian: Paralysis from Brain Disease.

corpus striatum and neighboring parts, with the exception of the heat and swelling referred to.

It was evident in this, as in other cases associated with this special symptom, that the lesion, whatever or wherever it was, exerted an inhibitory influence, either directly or reflexly, on the vaso-motor centre.

The pons varolii is regarded as especially a regulative centre for vaso-motor nerves, but the symptoms were manifestly not those that follow damage to that sensitive body. It was hardly possible to locate the lesion in the crus cerebri, as in such a case there would probably be a concurrent paralysis of the third nerve.

Injuries to the optic thalamus, however, owing to its close proximity to the principal vaso-motor centre, have so frequently been found associated with these symptoms of increased temperature and swelling, that in the case related, this part was believed to be seriously involved.

Of two cases that have fallen under my observation, where the lesion was supposed to involve both the pons varolii and the crus cerebri, I give here the history of one. The attack occurred suddenly in the person of a working man aged fifty-five, who, for years before had not been detained at home even for a day by reason of illness. The character of the paralysis relating to the extremities, the face, and the tongue was of the usual character. Beyond this, however, there was some difficulty of deglutition, with painful hyperæsthesia of the paralyzed side of the face, due probably to injury to the fifth nerve. The patient also became exceedingly emotional, bursting into tears at the slightest cause, and frequently without cause. These symptoms pointed undoubtedly to lesion of the pons in its upper lateral portion. The symptom that indicated damage to the crus cerebri was paralysis of the third nerve, on the same side as the brain lesion. The results of this condition are well known, being ptosis of the eyelid, dilatation of the pupil, and external squint. This last symptom is due to the fact that all the muscles of the eyeball become paralyzed, except the external rectus and superior oblique. The tempera-

ture of the paralyzed limbs was decidedly higher than normal, as has been frequently observed in demonstrated cases of lesions of the pons varolii.

In a speedily fatal case that I saw a few weeks ago, in consultation with Dr. A. R. Carman, in addition to the difficulty of deglutition, paralysis of the tongue, and other symptoms indicative of damage to the pons varolii, we noted a rare symptom, designated by the term "pulsus deferens." The pulse in the right radial artery would repeatedly disappear and reappear, and was suggestive as indicating injury to the vaso-motor nerve-tracts and consequent interference with the contractile power of the arterial walls.

Severe injuries to the pons in its central part give rise to profound coma with double hemiplegia. These cases are, as a rule, rapidly fatal. If the damage to this central part is slight, the patient may regain consciousness, but with a more or less marked bilateral paralysis. When the lower half of one lateral region is involved, the facial paralysis is on the side of the brain lesion, while the limbs of the opposite side are affected in the usual manner. It is readily understood that this so-called alternate paralysis occurs because the fibres of the facial are damaged below, instead of above their decussation in the pons, while the motor fibres supplying the extremities become involved above their decussation in the medulla. I have no record of any case in which this irregular complication existed.

The cortical substance of the brain is very frequently the seat of effusion, and the combinations of symptoms following damage to this part are many.

A very interesting case, in which I was led to believe that this was the damaged portion, came under my care a few weeks since in the person of a physician in an adjoining city. Four years ago, after suffering for some time from intense pain in the head, associated with a tendency to sleep, he became suddenly unconscious and fell to the ground. The resultant hemiplegia, at first incomplete, soon became complete. In five days he began gradually to regain the power of motion, until in this respect

he had quite recovered. At no time was there marked anæsthesia, and but very slight, if any, deviation of the tongue or face. The sphincters were at first relaxed, but power was speedily regained over these muscles. There was temporary ptosis, and also pronounced aphasia from the beginning.

Whether at the time of, or shortly after the attack there were tonic or clonic spasms of the limbs, symptoms often observed in lesions of the cortical gray matter, I did not ascertain. At the present time, four years after the attack, the patient presents the appearance of perfect health. There is no paralysis of any kind, and aphasia is noticeable only in an occasional inability to remember a word; but there exists marked amnesic defects in writing, and what is far more serious, he can understand hardly anything he reads, although it becomes perfectly plain when read to him. The character of the onset of the attack in this case, the final and somewhat rapid disappearance of the hemiplegia, the exemption of the face and tongue, the absence of anæsthesia, and the presence of aphasia, all pointed to the cortex as the location of the lesion.

Anæsthesia, it is unnecessary to say, is a very common symptom in hemiplegia, but occasionally we meet with irregular forms of it, where it is out of all proportion to the motor paralysis, and persists after the power of motion is fully regained. I have no records of this condition, but the post-mortems of Turck and the investigations of Veyssière "go to show that complete hemi-anæsthesia may be produced by a considerable lesion just outside of the optic thalamus, involving the peduncular expansion (internal capsule) just at the part where it begins to unfold into the foot of the radiating crown of Reil.

In Turck's cases, injuries in and about this region gave rise to a hemi-anæsthesia which persisted for many months, and even for one or two years after the motor paralysis had almost wholly disappeared." Unilateral hyperæsthesia, on the contrary, is somewhat more common, and in a case of especial interest, that I saw with Dr. J. W. Greene, it was a

prominent symptom. The patient was a cashier in one of our banks, and a hard drinker. His right side became suddenly and completely paralyzed, followed by such an acutely sensitive condition of the limbs, that the slighest touch caused great pain.

Taking into consideration the completeness of the paralysis, and the severity of the other symptoms, the case seemed about as unpromising as possible, yet in a few months recovery was approximate if not complete. The treatment was almost entirely electrical, by the methods of general faradization and central galvanization. I have frequently met him on Broadway, walking with all his former vigor. We do not positively know the cause of this symptom, but it has been suggested that as destruction of one of the sense-centres for common sensibility results in hemi-anæsthesia, so a neighboring lesion, resulting merely in a reflex irritation of this sense-centre, may be followed by hemi-hyperæsthesia.

In all the foregoing cases it is to be presumed that the pathological condition was either rupture or occlusion of vessels. I have made no attempt in this paper to discriminate between the two; indeed, it is no easy matter to do this in many cases, and the differential diagnosis in this respect is, by no means, so important as the regional diagnosis, which has mostly engaged my attention.

There is a third cause of hemiplegia, however, which we understand very little about, but which may be perhaps not quite so infrequent as is generally supposed. I refer to a spasm of the vessels. In the case that came under my observation the attacks were intermittent in character, resulting in partial hemiplegia, which would soon pass away. These attacks extended over several months, and finally resulted in the death of the patient. A post-mortem made at my request by Dr. Henry T. Pierce revealed various abnormal conditions of the brain, but neither rupture nor occlusion of any vessel, and rendered it probable that the cause was functional and spasmodic.* A point of considerable practical

* This case will be found in the New York Medical Journal, September, 1877.

interest in the consideration of this case lies in the fact that applications of electricity for the purpose of affecting the vaso-motor system resulted in an immediate cessation of these attacks, which continued from the 25th of September to the 4th of December, when a most violent attack carried him off.

The infrequency with which we meet serious impairment of the function of the nerves of special sense in cases of hemiplegia following brain lesions, renders the subject perhaps of secondary importance. The explanation for this exemption is very much the same as has been offered by Broadbent in the case of motor phenomena, and is perhaps even more conclusive; for the researches of Lockhart Clarke certainly show that the nuclei of the two optic and two auditory nerves respectively are very intimately connected. As a matter of fact we know that not only do ordinary lesions of one hemisphere fail to impair sight, but that almost complete destruction of one hemisphere may leave these functions intact, through the power of the opposite hemisphere to take cognizance of visual impressions. In regard to sight there are some exceptions to this rule of exemption in damage to the hemispheres, but none probably as regards hearing, since the nuclei of the two auditory nerves lie near the junction of the pons with the medulla, and hearing can hardly be impaired without there is either a lesion at this point, an injury near the origin of the nerve trunk, or an incomplete thrombosis of the basilar artery. Sight, on the other hand, may not only be occasionally affected by damage to one hemisphere, but the extent of the optic tract within the cranium, and its varied blood-supply, render it more liable to injury in any form of intracranial disease than the sense of hearing.

Various local lesions, resulting not only in hemiplegia, but also in total or partial blindness, might be enumerated; but I desire at this time to consider an occasional loss of this function which is only temporary and due probably to indirect or reflex effects of injuries to brain-substance in close proximity to the corpora quadrigemina.

As a parallel to the above we may have, as a result of an epileptic seizure, temporary paralysis of the limbs. One case of this kind, where the paraplegia was complete, and lasting for over twenty-four hours, I have myself seen. In the same way, then, either with or without an epileptic attack, we may have complete loss of sight. It is said that hemiplegia, when associated with this anomaly of temporary blindness, is apt to be preceded by epileptiform convulsions, and in the illustrative case which I have to relate, this was undoubtedly the mode of onset. This case came under my observation some years ago, in the practice of a Dr. William Miller, now deceased, who, although not a regular physician, had, in certain directions, an immense, and perhaps an unparalleled experience. Until within a few years since, I had never reported it,* from the fact that at the time it seemed to me to be entirely exceptional and unexplainable. In the light of subsequent experience, however, the result then noted would seem to be not altogether exceptional, nor devoid of the possibility of rational explanation.

Some years previous the patient, a man in middle life, had suffered from a convulsive seizure, which was, I suppose, epileptic in character, and these at long intervals had been repeated. Two months before he came under my observation, he was prostrated by an epileptiform attack of unusual severity, resulting not only in incomplete paralysis of one side of the body, but a condition also of total blindness. There was in this case a reversal in the order of recovery, for two months after the attack—while the arm had almost wholly regained its normal power, the strength of the leg was still impaired to a considerable degree. An induction current, as powerful as could be well borne, was applied simultaneously to both eyes of the patient for about two minutes, and was followed by an immediate restoration of sight. This result, surprising as it seemed at the time, and almost incredible as it may appear to those who have given the subject no thought, or

* Virginia Medical Monthly, September 18, 1878.

who have never heard of cases analogous, is, in truth, not so astonishing as it would seem, neither indicating any mysterious power nor novel therapeutic action of the current. Judging from reports of other cases, and the probability of the purely functional or indirect cause of the blindness, recovery would probably have taken place spontaneously, and was only hastened by the reflex stimulation of the electricity.

The length of time during which sight was lost in this instance would seem to be exceedingly rare, for among a number of cases mentioned by Bastian,* he refers to one in which the blindness "lasted for an unusual time—as long as six weeks." This patient, while in apparently perfect health, had several attacks of numbness in the left arm, which finally resulted in almost complete loss of power, and at the same time left him so totally blind that he was unable to see daylight.

The recovery of sight was rapid, and continued permanent. If we attempt to explain the interesting phenomena of the sudden and total loss of sight and its sudden recovery on the theory of indirect or reflex influences, some might suggest that, in the light of a better knowledge of cerebral physiology and pathology, these views are not received with the same favor as formerly. It must not be forgotten, however, that morbid anatomy is as yet far from being co-extensive with pathology. The most profound derangements of the functional activity of the nerve-centres may exist, and yet by none of our advanced methods of investigation has it been possible to discover any appreciable lesion; and until we have more accurate knowledge in this direction, the theory of reflex influences, on which we must fall back for explanation, although beyond the sphere of absolute verification, is, at all events, plausible and, to a certain extent, rational.

* Paralysis from Brain Lesions, p. 116.